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Cognitive function in major depression

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Summary

Forty patients with a major depressive episode were divided into equal endogenous and neurotic sub-groups using the Newcastle scale. They were all rated on the 17-item Hamilton scale and with a variety of neuropsychological tests. They were compared with 20 age- and education-matched control subjects. Both endogenous and neurotic groups had impaired memory function on the auditory verbal learning test; recall and recognition were equally impaired suggesting that effort was not a major determinant of performance. The endogenous group was more impaired on digit symbol substitution and the Trail making test (A and B). Impairment was correlated with symptom scores on the Hamilton and Newcastle scales, even after allowing for the effect of age. It is concluded that the conventional distinction between organic and functional impairment breaks down in severe depressive illness. The implications of this for clinical neuropsychological testing and the anatomy of the brain dysfunction in depressive illness are discussed.

Key words: Unipolar depressive illness; Cognitive function; Memory

Introduction

It would be highly unconventional to define depressive illness as anything other than a disorder of mood. Nevertheless, its reliable diagnosis requires the identification of other phenomena, most notably the so-called biological symptoms of depression. Usually ignored, at least for diagnostic purposes, is the frank impairment of cognitive function which also occurs in depressive illness. In some ways this is surprising because it is easier to measure memory impairment, for example, than it is to estimate sleep disturbance or weight loss with any accuracy. Central to renewed interest in this area is a renaissance of efforts to link theories of cognitive neuropsychology to the anatomy and physiology of underlying brain function. This is occurring as a primary theme of modern neuroscience. It is now relevant to psychiatry in particular because of the availability of

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functional neuroimaging techniques, either Single Photon Emission Tomography (SPET or SPECT), or Positron Emission Tomography (PET).

Early studies of neuropsychological performance in depressed patients indicated definite deficits in memory from complex tests of coding and subsequent retrieval of verbal material (Cronholm and Ottoson, 1961; Sternberg and Jarvik, 1976; Stromgren, 1977). These abnormalities were reversed with treatment of the depression. Caine (1986) has reviewed this and subsequent work and reflected on the relative paucity of comprehensive neuropsychological studies. For the most part recent work has been largely dominated by how mood might influence different components of memory performance. In consequence of this and, perhaps, because of the quite separate strands of interest generated by the so-called cognitive theory of depression (Beck 1963), specialized psychological constructs have tended to receive precedence over standard neuropsychological approaches to brain dysfunction. Indeed, the view has been commonly held that tests usually employed for the investigation of organic disorder are in some sense invalid for the investigation of functional illness. If the sole purpose of testing were the identification of organic disorder this would be correct, but not if, instead, we wish to employ neuropsychology to develop theories of functional neuroanatomy in non-organic psychiatric disorder.

The present study employed a short battery of neuropsychological measures in common use in other neuropsychiatric disorders. They were chosen to tap a range of neuropsychological domains and to provide scope for cross-diagnostic comparisons. An effort was made to recruit patients across the spectrum of unipolar depressive disorder. The unimodal distribution of depressive syndromes described by Kendell (1968) implies that division into neurotic and endogenous sub-groups is arbitrary. However, the grouping of patients at either end of this continuum by use of the Newcastle scale can be justified by their different response to ECT (Carney et al., 1965), different rates of cortisol suppression after dexamethasone administration (Carroll et al., 1981) and their different long-term prognosis (Lee and Murray 1988). It is still useful to employ neurotic and endogenous sub-groups for the purposes of comparison with control or other patient groups.

The aims of this study were three-fold. Firstly to replicate and extend earlier studies examining memory, concentration and psychomotor speed in depressed patients by means of a matched-control study. Secondly to test the hypothesis that neuropsychological deficits will differentiate between the neurotic and endogenous sub-groups of depressed patients; endogenous patients would be expected to show greater impairment. Thirdly to assess how closely these neuropsychological deficits correlate with conventional estimates of clinical severity across the whole spectrum of the depressed patient sample. The relationship of these results to SPET examination of these patients will be the subject of another report.

Methods

Subjects

Forty patients (21–67 years) seen at the Royal Edinburgh Hospital and fulfilling DSM-III-R criteria for major depressive episode with or without melancholic or mood-congruent psychotic features were entered in the study. The Schedule for Affective Disorders and Schizophrenia (SADS) was used as a standardised diagnostic interview (Endicott and Spitzer, 1978) and severity of depression was assessed by means of the 17-item Hamilton Depression Rating Scale (Hamilton, 1960). Patients with a bipolar illness were not included in the study. Patients were required to be in good physical health and have no associated psychiatric diagnosis, in particular, substance abuse. Whilst we attempted to recruit drug-free patients, nineteen who had been on a stable dose of an antidepressant and/or hypnotic for 2 weeks or more at the time of assessment were also accepted. The antidepressant drugs being taken by the patients were as follows: Amitriptyline (10), Fluoxetine (4), Clomipramine (2), Imipramine (1), Mianserin (1), Tranylcypromine plus L-Tryptophan (1). Patients who had taken an overdose were studied at least one week after the incident.

Patients who had been treated with ECT within six months, were excluded. Patients with 'endogenous' and 'neurotic' depression were distin-

guished by the Newcastle Depression Scale (Carney et al., 1965) with a cut-off score of 6. As far as possible suitable consecutive admissions with major depression were studied. However, over the duration of the study 15 patients fulfilled the criteria for inclusion but were not entered; testing was precluded in five cases because of the severity of their agitation, eight were omitted because of changes made in their medication before testing could be performed and two refused to participate.

The control group consisted of 20 subjects recruited from hospital staff and local volunteer groups. These were free of psychiatric and physical illness and matched for age, sex and years of education. All subjects were tested under standardised conditions.

Neuropsychological testing

1. National Adult Reading Test (NART, Nelson and O'Connell, 1978): a reliable estimate of pre-morbid IQ in demented patients which is also being investigated in patients with functional psychiatric disorders (Crawford et al., 1987).

2. Auditory Verbal Learning Test (AVLT: Rey, 1964, Lezak, 1983), a test of new learning ability and immediate and short-term memory, including recognition. Subjects are presented with a 15 item list which has to be recalled immediately after 5 consecutive repetitions. Delayed recall and recognition of the same list are tested after 30 min. The AVLT has been suggested to be particularly appropriate to testing in depressed patients, because memory assessment by repeated presentation minimizes the effect of poor attention span (Sweeney et al., 1989).

3. Wechsler Adult Intelligence Scale-Revised (Wechsler, 1981) subtests (WAIS-R): a) Digit Symbol Substitution Test: This is a time limited test of coding and psychomotor speed. b) Block design: a test of visuo-spatial and constructional ability which is also time limited.

4. Digit span forwards and backwards: a test of concentration and immediate recall. This was presented and scored as recommended in the Wechsler Memory Scale-Revised (Wechsler, 1987).

5. Trail-making A and B (Army Individual Test Battery, 1944): both test attention, concentration and psychomotor speed, with Trails B also requiring the ability to shift cognitive set. In the case of Trails B, some patients had such difficulty completing the test that a maximum time of 5 min (300 s) was allowed.

6. Verbal Fluency (Borkowski et al., 1967); this task appears to test the integrity of frontal lobe function by requiring the generation of as

TABLE 1

Ratios, means and standard deviations for baseline demographic and clinical variables with values for Chi-square, F ratios (one way ANOVA), unpaired *t*-test and associated *P* values for the cross-sectional tests

	Endogenous (20)	Neurotic (20)	Control (20)	F Ratio, chi-square	*P* value
Sex (M/F) ratio	11:9	6:14	8:12	$\chi^2 = 2.6$	NS
Socio Economic Class (I+II:III:IV+V)	6:9:5	3:13:4	11:7:2	$\chi^2 = 8.1$	0.09
NART IQ, mean(SD)	107.0 (14.2)	104.7 (14.1)	111.7 (10.0)	1.55	NS
Age, mean(SD)	48.6 (14.2)	44.4 (12.7)	47.0 (16.1)	0.43	NS
range	24–67	22–60	23–65		
Education, mean(SD)	13.2 (4.2)	12.1 (3.0)	13.3 (3.3)	0.71	NS
range	9–22	9–20	10–18		
Antidepressants	11	8	NONE	$\chi^2 = 0.9$	NS
HDRS, mean(SD)	24.1 (5.8)	21.3 (4.7)	N.D.	$t = 1.65$	0.10

many words beginning with the letters F, A and R as possible in 3 sixty second periods.

Statistics

Patients were grouped by clinical diagnosis as neurotic or endogenous using a cut-off of 6 on the Newcastle scale (Carney et al., 1965). The comparability of the patient groups with controls was examined for age, NART IQ, education, handedness, sex and social class using the appropriate cross-sectional statistic (one way ANOVA or chi-square). The responses on the five repeated trials of the AVLT were compared using repeated measures ANOVA. The other neuropsychological test scores were examined by one way ANOVA. Differences between groups for individual measures were identified post hoc with Scheffe's test. Test scores for the 40 patients were then subjected to a correlational analysis. All calculations were performed using SPSS for the Apple MacIntosh.

Results

The three groups (control, neurotic and endogenous) were well matched for age, years of education, handedness and gender (Table 1). The volunteers showed a slight excess of social class I and II occupations; this is likely to have arisen from the generic occupational term of nurse leading to over-classification of some volunteers. The closely similar NART IQ estimates provide further validation of this test as an estimator of premorbid intellectual function in depressive illness (cf. Crawford et al., 1987). Depressive symptoms are summarised by the Hamilton rating scale and the Newcastle scale. Within the depressed groups, 48% were taking an antidepressant at the time of testing, none were taking a neuroleptic (this was an exclusion criterion) and 15% were taking a short-acting benzodiazepine at night. There was no significant difference between the depressed groups in relation to current drug exposure (see Methods for further details).

The results for the five repeated trials of AVLT are illustrated in Fig. 1. Trial 1 represents a measure of immediate recall, improvement on subsequent trials reflects learning. Both neurotic and endogenous patients were impaired on all trials and to a similar degree (Fig. 1). The slopes of the curves were not significantly different. The result is summarized as the sum of all 5 trials in Table 2 together with evidence that patients were

TABLE 2

Mean (standard deviation) for total number of words recalled across the 5 AVLT trials, delayed recall and recognition at 30 min, and an index of forgetting (trial 5 score minus delayed recall), raw scores for the WAIS-R digit span forwards and backwards, block design and digit symbol substitution (DSST), average number of words generated over 3 min beginning with the letters F, A, R (verbal fluency) and number of seconds taken to perform Trails A & B

	Endogenous (20)	Neurotic (20)	Control (20)	*P* value for F test	Scheffe ($P < 0.05$)
Sum 5 AVLT Trials	40.5 (12.0)	44.0 (8.2)	55.8 (11.2)	0.0001	C > N, E
Delayed Recall	8.0 (3.5)	9.2 (4.0)	12.2 (2.9)	0.002	C > N, E
Recognition	9.2 (3.6)	9.4 (6.3)	13.4 (1.9)	0.004	C > N, E
'Forgetting'	2.3 (1.7)	2.4 (2.3)	1.5 (1.5)	0.21	
Digitspan (forward)	9.3 (2.0)	8.8 (1.7)	9.2 (1.9)	0.66	
Digitspan (back)	7.0 (2.2)	6.4 (2.1)	7.8 (2.2)	0.19	
Block design	23.9 (12.8)	29.8 (10.2)	32.1 (11.1)	0.12	
DSST	37.9 (15.0)	45.9 (12.8)	53.7 (10.6)	0.001	C > E
Trails A (secs)	50.7 (24.5)	40.9 (10.1)	35.8 (11.9)	0.02	C < E
Trails B (secs)	144 (85.1)	100.3 (70.3)	68.9 (22.5)	0.002	C < E
Verbal fluency (Words/min.)	12.8 (5.8)	13.2 (6.3)	15.8 (4.8)	0.23	

Scores for the three groups were compared by one way ANOVA. Differences between groups were located post hoc with the Scheffe test; abbreviations are, C = Control group, N = Neurotic group, E = Endogenous group.

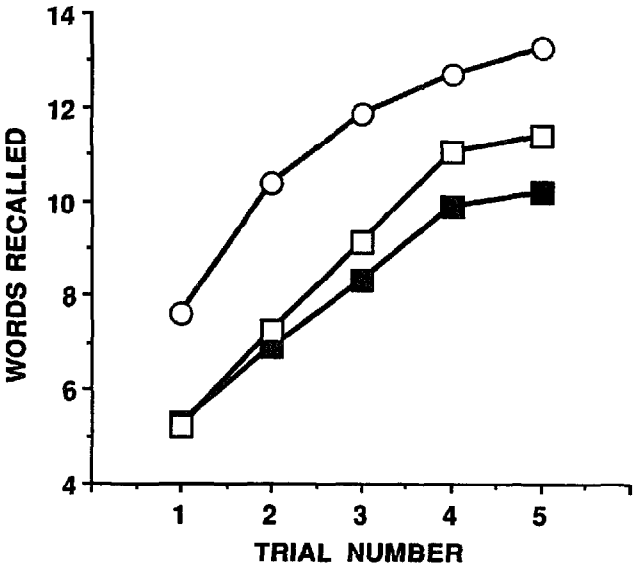

Fig. 1. The mean number of words recalled on successive trials of the AVLT by control (open circles), neurotic (open squares) and psychotic (closed squares) groups. Repeated measures ANOVA showed a main effect of group ($F(2, 57) = 12.1$, $P = 0.000$). The patient groups were clearly different from controls but not from each other. Further details in text and Table 2.

also impaired on delayed recall at 30 min and recognition of previously learned words at 30 min but not for an index of 'forgetting' (trial 5 recall-delayed recall).

Table 2 also shows the means, standard deviations, F test summary and post-hoc Scheffe comparison for the other neuropsychological tests. Scores represent number of correct responses so that impairment will be shown as a reduced value, except for Trails A and B where results are given as number of seconds taken to complete the test and impairment is expressed as an increase in this time.

There are significant differences between the 'endogenous' patients and the control group on three of the time-dependent tests: Trails A, Trails B and digit symbol substitution (DSST). Neurotic patients were less impaired on these tests. Block design, digit span (forwards and backwards) and verbal fluency (VF) do not show statistically discernible differences across the groups.

The association between impaired test performance and clinical variables was examined within the forty depressed patients. The correlation be-

TABLE 3

Partial correlation between neuropsychological test scores and clinical variables in 40 patients controlling for age; *, $P < 0.05$, **, $P < 0.01$, *** $P < 0.001$

	AVLT 5	Delayed recall	Recognit'n	Sum AVLT trials	DSST	Trails A	Trails B	Hamilton	Newcas	Retard'n	Medicat'n
AVLT 1	0.32 *	0.40 **	0.24	0.55 ***	0.39 **	0.04	−0.17	−0.03	−0.04	−0.10	−0.08
AVLT 5		0.82 ***	0.66 ***	0.88 ***	0.39 **	−0.25	−0.42 **	−0.34 **	−0.36 **	−0.10	−0.06
Delayed recall			0.76 ***	0.83 ***	0.44 **	−0.08	−0.34 **	−0.28 *	−0.34 **	−0.02	−0.16
Recognit'n				0.63 ***	0.39 **	−0.27 *	−0.24	−0.17	−0.17	−0.14	−0.20
Sum AVLT trials					0.51 ***	−0.22	−0.48 **	−0.36 **	−0.37 **	−0.11	−0.16
DSST						−0.43 **	−0.50 **	−0.42 **	−0.50 **	−0.34 **	−0.09
Trails A							0.44 **	0.31 *	0.30 *	0.29 *	−0.11
Trails B								0.43 **	0.42 **	0.23	0.09
Hamilton									0.52 ***	0.05	0.07
Newcastle										0.52 ***	0.27 *

tween the neuropsychological tests showing group differences and the clinical variables are shown in Table 3; the associations are controlled for the effects of age. It will be obvious that the neuropsychological abnormalities are moderately correlated with each other. This limits the value of comparing the correlation coefficients derived with the Hamilton and Newcastle scales for different tests. However, it is obvious that increased depression ratings are associated with worse performance on most tests (Table 3). The only exceptions are performance on the first trial of AVLT (AV1) and on recognition memory. The DSST appears to be the most sensitive to the range of symptom scores exhibited within the sample and can account for 25% of the variance in the clinical ratings. A more detailed examination of subscales of the Hamilton and Newcastle scales suggested that impairment of performance on the time dependent tasks, but not memory performance, was related to clinically evident psychomotor retardation (Table 3).

There was no association beyond chance between cognitive function and clinical variables such as a history of familial affective disorder or personal history of attempted suicide (not illustrated). The ongoing consumption of antidepressant drugs was also without effect upon cognitive function (Table 3).

Discussion

The present results support and extend previous studies in which neuropsychological dysfunction has been described in depressed patients. Our sample was comprised of patients showing high levels of symptoms. This was true of both neurotic and endogenous groups so that the mean Hamilton scores were comparable. They were not identical however, and the shared variance between Hamilton ratings and Newcastle score means that it is not possible to distinguish unambiguously between the effects of illness severity and the presence of particular endogenous symptoms such as retardation. Previous factor analytic studies of the Hamilton rating scale have also, depending on the particular sample used, suggested a subsidiary factor related to endogenous symptoms (Thompson, 1989).

Our findings relate primarily to deficits in memory and speed-related visuo-spatial motor tasks. Impairment of memory on the Auditory Verbal Learning Test was observed for all trials and was similar for delayed and recognition memory. Our findings support those of Golinkoff and Sweeney (1989), who also described recognition deficits in depression. They made the further point that memory impairment was not simply related to task difficulty; thus, recall is a more effortful task than recognition yet both appear to be equally impaired. It had been suggested in the past that the poor performance of depressed patients reflected lack of effort or motivation (Cohen et al., 1982). Richards and Ruff (1989) explicitly excluded motivation as an important determinant of performance in depression. In the present study total learning capacity and the ability to retrieve material unaided were also impaired. The rate of 'forgetting' was similar in patients and controls. The magnitude of the impairment in memory function was similar in both neurotic and endogenously depressed patients, although correlational analysis indicated that there is a general relationship between severity of symptoms and impairment of function across the whole group of depressed patients. Our findings in relation to memory tests are also consistent with those of Wolfe et al. (1987) who compared performance on the AVLT and Verbal Fluency in 20 unipolar depressed patients with 20 controls matched for age and years of education. The latter study described worse memory performance in bipolar patients. In contrast, Sweeney et al., (1989) found that severity of depression was only significantly correlated with performance on AV1 of the AVLT in 21 elderly patients; learning across the 5 trials and delayed recognition were not significantly impaired when subject's scores were compared to normative data. The absence of matched controls limits the interpretation of this study, but, in any case, the finding that elderly patients are less cognitively impaired than younger patients by depressive illness flies in the face of clinical experience and indeed other studies (see Jorm, 1986). Abas et al., (1990) showed both memory and motor impairment in elderly depressed subjects which, interestingly, were clearly related to CT scan changes and not to the sever-

ity of depressive symptoms. The latter finding and the failure to show full reversal of cognitive deficits with treatment of the depression, highlight that different factors, related to aging and the possible onset of Alzheimer type dementia, are prominent in determining the profile in the elderly.

Performance on Trails A and B and DSST was most impaired in our endogenous group. This appears to reflect decreased psychomotor speed in more severely depressed patients. Thus, there is a strong correlation between illness severity as measured by the Newcastle scale and the rating of retardation. It may be of significance that performance on block design was within the normal range. Block design is a complex visuo-spatial constructional task with a component of motor speed but this appears to influence total performance less than in the other tests.

The simplest interpretation of the present findings is that worsening depressive illness progressively impairs cognitive function, with memory tasks the most and visuomotor speed the next most vulnerable to its effect. These findings are not explained by a global attentional difficulty since performance on digit span was virtually unaffected. Previous findings on digit span in drug free depressed out-patients have been similar (Richards and Ruff, 1989) as have those employing a purer attentional test of simultaneous matching to sample in the elderly (Abas et al., 1990).

Any coherent explanation of the brain mechanisms involved in depression must explain these phenomena. The memory impairment of depressed patients is particularly striking and occurs in both neurotic and endogenous groups. There is prominent involvement of either hippocampus or medial diencephalic nuclei in the genesis of amnesia in patients with brain lesions (Squire, 1987). These areas must be candidates for the locus of the effect in depression also. The hippocampus appears the most likely because disruption of diencephalic function, as probably occurs in Korsakoff's psychosis, produces significant frontal lobe symptoms. The mechanism must allow recovery and could accordingly result from reversible but persisting changes in neurotransmitter function. The effects of particular neurotransmitters in the hippocampus are poorly understood at a functional level. However, an involvement of 5-hydroxytryptamine or acetylcholine, both of which are implicated in the neuropharmacology of affective illness, may be worth investigating since both have dense projections to hippocampal areas.

The involvement of sub-cortical mechanisms or limbic structures is also supported by cross-diagnostic comparison with other diseases having a defined sub-cortical pathology such as Huntington's disease, Parkinson's disease or even HIV infection. They have a similar, if by no means identical, neuropsychological profile (Cumming and Benson, 1988). These illnesses can, interestingly, be associated with depressive symptoms and in each case, one focus of the basic pathology appears to be in the basal ganglia. The retardation of severely depressed patients demands a mechanism additional to that which would explain uncomplicated amnesia and basal ganglia dysfunction could be involved. It is likely that sub-cortical centres such as the basal ganglia and thalamus are functional bottlenecks where changes in the efficiency of a variety of afferent or efferent pathways may produce slowing and cognitive impairment. However, impairment of function in structures subserving loops to and from neocortical areas might be expected to impair function in those areas as well. It is interesting, therefore, that in our younger patients the normal performance on verbal fluency, a special example of search and retrieval from long term verbal memory, argues against an important frontal component whilst the performance on block design is against the involvement of non-verbal constructional strategies requiring parietal and frontal neocortex. In general, the preservation of tasks subserved by cortical structures distinguishes the depressive syndrome from Alzheimer type dementia. It may be evident that we prefer to place emphasis upon neuropsychological deficits as clues to the underlying brain abnormalities in depressive illness. This is against much of the prevailing theory in relation to 'depression' which assumes psychological explanations to be sufficient to account for cognitive impairment. On this basis, learned helplessness or cognitive interference (e.g. see Miller, 1975)

could, no doubt, be made to encompass these results.

About half of our patients were taking antidepressant drugs but this appears not to have influenced our findings. The effects of antidepressant drugs upon cognitive function have been reviewed recently by Thompson (1991). There have been almost no studies in volunteers to examine the effects of antidepressants given at clinically relevant doses for periods of time beyond two weeks. Acute effects appear attributable to sedative actions and these wear off in 1–2 weeks. Most of the work in patients appears often to have confused the consequences of depressive illness itself with the effects of acute administration of sedative tricyclics. In general, prolonged antidepressant treatment tends to improve cognitive function as depressive symptoms remit. This is true for drugs and indeed, for Electroconvulsive therapy (Frith et al., 1984), despite its reputation for causing memory impairment. The decision to include patients on stable doses of drugs allowed us to study a more representative sample of seriously ill patients. At presentation, patients were often receiving sub-therapeutic doses of antidepressants or they had failed to respond to conventional doses. In many cases, the withdrawal and necessary 'washout' of the current antidepressant would have been difficult to defend on clinical grounds either because the patient required an increase in dose or a change in treatment without undue delay. In any case, the withdrawal of an antidepressant may have prolonged neuropharmacological consequences potentially as confusing as those of any continuing treatment to which the patient is habituated.

The present findings are highly relevant to the differential diagnosis of dementia in individual psychiatric patients. Most of our depressed patients scored more than one S.D. below the control means in memory tests, and half of the endogenous patients were more than two S.D. longer than the control mean to complete Trails B. The results are summarised in Table 4. The findings in individual cases would often be sufficient to raise the suspicion of dementia. For example, in relation to individual tests, Lezak (1983) has commented that the DSST is 'consistently more sensitive to brain damage' than other Wechsler subtests. However, in depression DSST performance is highly correlated with the Newcastle score. The present findings show that the implied dichotomy between organic and functional illness breaks down when a functional illness is severe. Furthermore, the test abnormalities may point to the underlying neural substrate of the disorder.

TABLE 4

Percentages of sample of 20 endogenous and 20 neurotic patients showing performance deficits one or two standard deviations from the mean (1 SD, 2SD) for neuropsychological tests

	1 S.D.		2 S.D.	
	Endogenous	Neurotic	Endogenous	Neurotic
NART IQ	30%	35%	10%	5%
Sum 5 AVLT Trials	75%	55%	30%	5%
Delayed recall	70%	60%	40%	25%
Recognition	75%	65%	45%	30%
DSST	75%	50%	30%	15%
Trails A	45%	20%	30%	5%
Trails B	70%	35%	50%	15%

Results also shown in Table 2, where further details are included in the legend.

It is now difficult to see why the measurement of depression should not include estimates of cognitive impairment. While such estimates are unlikely to be more specific than other symptoms such as sleep disturbance, or weight loss, that receive emphasis in existing scales, they may be more easy to measure accurately and they may be significant in assessing response to treatment and, indeed, predicting outcome. Together with such a purely empirical advantage it is possible that a greater emphasis on neuropsychological function may assist the transition for psychiatrists from thinking about illnesses in purely phenomenological terms to thinking about them in terms of regional cerebral dysfunction.

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